



Jian Xiong

Postdoctoral Scholar, Chemical Engineering

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I thrive to understand the roles of lysosomes in physiological and pathological conditions. Lysosomes are both degradation compartment and metabolic controlling hub, and dysregulation of lysosomal functions are frequently implicated in a vast number of diseases including neurodegenerative diseases, however, the systematic knowledge of the molecular mechanism by which lysosomal contributes to these diseases is lacking. Ion channels are the primary mediators of neuronal activity, defects in neuronal ion channel activity are linked with many kinds of neurodegenerative diseases. Interestingly, besides typical ion channels that are involved in the neuronal activity, defects in lysosomal ion channels, such as TRPML1, CLN7 and CLC-7 are also implicated in neuropathy. My previous work as Ph.D student in University of Texas MD Anderson Cancer Center focused on regulation of lysosomal function by ion channels and metabolites. I discovered a mechanism of lysosomal Na⁺ channel regulate mTORC1 activation by regulating lysosomal amino acid accumulation. I also discovered role of glutamine in controlling lysosomal degradation capacity. In the meantime, I developed novel methods to isolate organelles. My ultimate research goal is to understand the key developmental pathways and how alterations in gene sequences and expression contribute to human disease, therefore, I am pursuing independent academic researcher as my career goal. Starting Feb 2022, I work with Dr. Monther Abu-Remaileh at Stanford University on role of lysosomes in neurodegenerative diseases. I use genetics, chemical biology and omics approaches to study lysosome function under various physiological and pathological conditions, especially age-associated neurodegenerative disorders, and monogenic neurodegenerative lysosome storage diseases. In Stanford, I aim to integrate ionic regulation, metabolomic regulation and functional proteomic regulation to systematically understand the biology of lysosome in physiological conditions and pathological conditions.

INSTITUTE AFFILIATIONS

- Member, Maternal & Child Health Research Institute (MCHRI)

PROFESSIONAL EDUCATION

- B.S., Wuhan University , Biological Science and Biotechnology (2010)
- M.S., University of Texas MD Anderson Cancer Center , Cell and Regulatory Biology (2014)
- Ph.D, University of Texas MD Anderson Cancer Center , Biochemistry and Cell Biology (2020)

STANFORD ADVISORS

- Monther Abu-Remaileh, Postdoctoral Faculty Sponsor
- Monther Abu-Remaileh, Postdoctoral Research Mentor

Publications

PUBLICATIONS

- **Glutamine Produces Ammonium to Tune Lysosomal pH and Regulate Lysosomal Function** *CELLS*

- Xiong, J., Luu, T., Venkatachalam, K., Du, G., Zhu, M. X.
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 - **Ameliorating cancer cachexia by inhibiting cancer cell release of Hsp70 and Hsp90 with omeprazole.** *Journal of cachexia, sarcopenia and muscle*
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 - **Regulation of lysosomal ion homeostasis by channels and transporters.** *Science China. Life sciences*
Xiong, J., Zhu, M. X.
2016; 59 (8): 777-91
 - **A gain-of-function TPC2 variant R210C increases affinity to PI(3,5)P2 and causes lysosome acidification and hypopigmentation.** *Nature communications*
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 - **Pharmacological Validation of ASIC1a as a Druggable Target for Neuroprotection in Cerebral Ischemia Using an Intravenously Available Small Molecule Inhibitor.** *Frontiers in pharmacology*
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 - **CAMK2/CaMKII activates MLKL in short-term starvation to facilitate autophagic flux** *AUTOPHAGY*
Zhan, Q., Jeon, J., Li, Y., Huang, Y., Xiong, J., Wang, Q., Xu, T., Li, Y., Ji, F., Du, G., Zhu, M. X.
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 - **mTORC1 controls lysosomal Ca²⁺ release through the two-pore channel TPC2.** *Science signaling*
Ogunbayo, O. A., Duan, J., Xiong, J., Wang, Q., Feng, X., Ma, J., Zhu, M. X., Evans, A. M.
2018; 11 (525)
 - **Loss of Smooth Muscle α -Actin Leads to NF- κ B-Dependent Increased Sensitivity to Angiotensin II in Smooth Muscle Cells and Aortic Enlargement.** *Circulation research*
Chen, J., Peters, A., Papke, C. L., Villamizar, C., Ringuette, L. J., Cao, J., Wang, S., Ma, S., Gong, L., Byanova, K. L., Xiong, J., Zhu, M. X., Madonna, et al
2017; 120 (12): 1903-1915
 - **Critical roles of Gi/o proteins and phospholipase C- β 1 in the activation of receptor-operated TRPC4 channels.** *Proceedings of the National Academy of Sciences of the United States of America*
Thakur, D. P., Tian, J. B., Jeon, J., Xiong, J., Huang, Y., Flockerzi, V., Zhu, M. X.
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 - **Differential mechanisms of action of the mucolipin synthetic agonist, ML-SA1, on insect TRPML and mammalian TRPML1.** *Cell calcium*
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2014; 56 (6): 446-56
 - **Bimodal voltage dependence of TRPA1: mutations of a key pore helix residue reveal strong intrinsic voltage-dependent inactivation.** *Pflügers Archiv : European journal of physiology*
Wan, X., Lu, Y., Chen, X., Xiong, J., Zhou, Y., Li, P., Xia, B., Li, M., Zhu, M. X., Gao, Z.
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 - **Drosophila TRPML forms PI(3,5)P2-activated cation channels in both endolysosomes and plasma membrane.** *The Journal of biological chemistry*
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